Ventilation Modes and Monitoring

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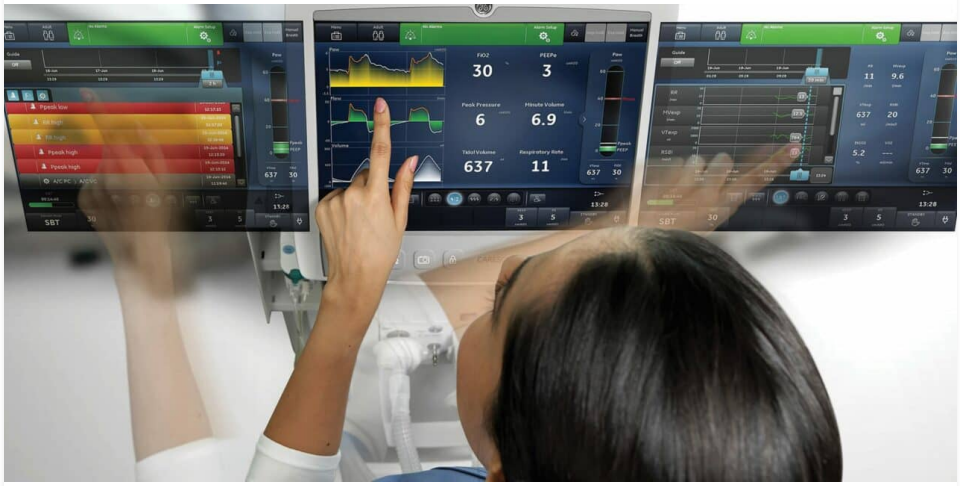

Management of mechanical ventilation requires selection of the appropriate mode, careful patient monitoring, and observation for possible complications.

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Whether it is used for airway protection, hemodynamic control, or the correction of ventilation or oxygenation abnormalities, mechanical ventilation is an important part of the tool kit used to care for critically ill patients.

Modes of Ventilation

Positive-pressure ventilators can be either volume cycled or pressure cycled. A volume-cycled ventilator delivers a set tidal volume, while a pressure-cycled ventilator delivers whatever tidal volume is needed to reach a set peak inspiratory pressure (PIP). Exhalation, of course, is passive, no matter what ventilation mode is used. There are roles for both volume and pressure ventilation. In general, volume ventilation is more common, but there may be a trend favoring pressure ventilation.[1,2]

There are five common varieties of volume ventilation: controlled mechanical ventilation (CMV), assist/control mode ventilation (ACMV), intermittent mandatory ventilation (IMV), synchronized intermittent mandatory ventilation (SIMV), and pressure-regulated volume-control (PRVC). CMV is the simplest form of mechanical ventilation. A set tidal volume is delivered at a set respiratory rate, with the patient unable to trigger the ventilator or to inspire gas freely through the airway circuit. Accordingly, only an apneic or paralyzed and sedated patient would be ventilated using CMV.[1,3]

ACMV still delivers a set tidal volume at a set respiratory rate, but also responds to a patient's inspiration. A spontaneous inspiratory effort triggers a full–tidal-volume breath and resets the timer for the subsequent mechanical breath. A patient breathing at more than the set ACMV rate has a higher work of breathing and is at risk for a respiratory alkalosis due to inappropriate hyperventilation because of a higher-than-anticipated minute ventilation. Often, patients who are awake do not tolerate ACMV well, and patients with expiratory obstruction are at risk for worsening overdistension.[1,3,4]

PRVC is a version of ACMV. Either the ventilator or the patient can initiate breaths, and a constant pressure, like that of pressure-control ventilation (PCV), is applied throughout both mechanical and patient breaths. The ventilator monitors each breath, comparing the delivered tidal volume with the set tidal volume and then adjusting inspiratory pressure to achieve delivery of the set tidal volume. If the delivered tidal volume is too low, the inspiratory pressure of the next breath is increased. If it is too high, the pressure of the subsequent breath is decreased. The advantage is a continuous response to changes in airway compliance and resistance, but the disadvantages can be hyperventilation or hypoventilation when there are changes in lung resistance or compliance.[4,5]

IMV combines CMV and spontaneous breathing. As with CMV, a set respiratory rate is delivered, regardless of the patient's respiratory efforts. A spontaneous breath, however, pulls gas from the ventilator circuit at whatever tidal volume the patient generates. The

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advantage of IMV is the patient has some control over ventilation and can tolerate ventilation without paralysis.[1,4]

Similarly, SIMV combines ACMV and patient effort with spontaneous breathing.[2,4] A background ACMV rate and tidal volume are set, and spontaneous breaths are given at the tidal volume generated by the patient. In addition, mechanical ventilation is synchronized to prevent a mechanical breath from being stacked (delivered at the same time as a spontaneous breath). If a patient is breathing at more than the SIMV rate, the patient controls both rate and tidal volume.[1-6] SIMV decreases both mean intrapleural pressure and PIP, compared with IMV, but has not been shown to affect cardiovascular variables significantly.[1]

With volume ventilation, ventilator parameters other than rate, tidal volume, and oxygen content can also be controlled. Most commonly, positive end-expiratory pressure (PEEP) is added to improve oxygenation through an increase in the functional residual capacity, pressure-support ventilation (PSV) is used to augment spontaneous breaths by partially unloading ventilatory muscles to decrease work of breathing, and inspiratory flow rate and the inspiratory-to-expiratory (I:E) ratio are changed to affect pressure and oxygenation.[1,4]

In PCV, pressure is the controlled parameter and time signals the end of inspiration. With PCV, the ventilator rapidly achieves a set pressure level and maintains it throughout the inspiratory time. The ventilator delivers whatever flow is needed to maintain that pressure level, so tidal volumes vary according to the patient's lung compliance. Accordingly, PIP is set to achieve a goal tidal volume, unless the patient is to be allowed to become hypercapnic in the interest of limiting PIP.[6]

PSV can be used as a full mode of ventilation (high-level PSV), as well as to support volume ventilation (low-level PSV). In high-level PSV, the patient controls all aspects of breathing except a set pressure limit. When triggered by a spontaneous breath, the ventilator delivers flow up to a set pressure limit, and flow continues until tidal volume demand decreases to a certain percentage of peak inspiratory flow. Tidal volumes vary as they do in normal breathing, and there is no guaranteed respiratory rate. PEEP can be added to both PCV and PSV.[1,3,4,6]

Airway pressure-release ventilation (APRV) uses positive airway pressure to augment spontaneous inspiration (reducing the work of breathing), then reduces the level of positive pressure to allow exhalation. This unrestricted, spontaneous ventilation reduces the likelihood of barotrauma or cardiac-function impairment. There is no set rate, so APRV is inappropriate for patients who are at risk for an inadequate spontaneous respiratory rate. This mode is particularly good for patients with obstructive lung disease.[1,3,4,6]

The choice of ventilation mode is based on the clinical situation, and there are no clear advantages associated with volume-regulated or pressure-regulated ventilation. Ventilator management choices, however, are important. Positive-pressure ventilation has been used since the 1950s, yet it was only in 1974 that clinicians recognized that it could actually induce lung injury.[7] Patient mortality may be affected by the choices made for mechanical ventilation.[2,7,8] The medical literature has recently focused on the need to prevent ventilator-associated lung injury (VALI), also called barotrauma or volutrauma. VALI is believed to arise from a number of sources. Cyclic lung distension by positive pressure can produce edema, bacterial translocation, and local and systemic inflammatory responses.[7] Ventilation using tidal volumes of 10 mL/kg or more frequently maintains clinical parameters reflected as arterial blood gas testing results. In a patient with lung injury, however, the ventilated lung volume is decreased, and the larger tidal volume can result in overdistension of the alveoli that are aerated. This overdistension is associated with lung injury from inflammation, increased vascular permeability, and loss of surfactant function.[2,7,8] While it would seem that low-pressure, low-volume ventilation would be a solution, VALI is also associated with the ventilation of injured lungs using low volumes and pressures. The mechanism for this injury is not well understood, but may be related to mechanical injury to lower airway segments, impaired alveolar function, and the effects of atelectasis.[2,7] In light of this, lung-protective ventilation strategies are being investigated. Attempting to address both physiologic ends of the VALI spectrum generally results in the suggestion of lower tidal volumes (usually 6 to 8 mL/kg), limit peak airway pressures (often to around 40 cm H2O), and use PEEP to facilitate oxygenation and maintain alveolar recruitment. Scientific evidence[1-3,7,8] available to date supports the use of these strategies, but not unequivocally. Clinicians making decisions about mechanical ventilation face an array of ventilator options and guidelines. It is important that they stay aware of the developing clinical evidence, make patient-specific decisions, and resist the temptation to make cosmetic changes that improve monitor or blood gas numbers, but do not serve patients' greater needs.[7]

Ventilator management is a multidisciplinary effort. Decreased complications and shortened durations for mechanical ventilation have been repeatedly demonstrated when nurses, physicians, and RTs are all empowered for patient management. The use of standardized protocols and standing orders also benefits patients. These decreases in complications and ventilator days naturally decrease expenses, which is particularly important in the current health care environment.[9-11]

Patient Monitoring

Mechanically ventilated patients require constant monitoring to evaluate changing airway dynamics. The most important parameters to track are PIP, exhaled tidal volume, and minute ventilation. Alarms

for these parameters should be thoughtfully set on the ventilator and, if the alarm sounds, it should be investigated by a critically thinking provider, not just silenced.

PIP varies with lung compliance and airway resistance, so changes can reflect patient improvement or serious problems. Increased PIP may indicate endotracheal tube occlusion, pneumothorax, increasing bronchospasm, or pulmonary edema. High peak pressures are clearly associated with VALI. Decreased PIP can indicate resolving airway obstruction or edema, but can also be due to inadequate volume delivery because of a ventilator-circuit leak or disconnection, unplanned extubation, or an insufficient gas supply.[1,3,4,6]

Exhaled volume evaluates both spontaneous and mechanical ventilation. The exhaled volume of a spontaneous breath indicates respiratory effectiveness, as a spontaneous tidal volume of at least 5 mL/kg is one parameter in the pre-extubation patient assessment. An exhaled tidal volume much lower than the set tidal volume indicates a loss of ventilator-circuit integrity or an air leak around the endotracheal tube. This can be from an inadequate cuff seal in an adult patient, or from an inappropriately sized cuffless endotracheal tube with too great an air leak in a pediatric patient.[1,3,4,6] A low exhaled tidal volume can also indicate an inadequate expiratory time due to obstructive lung disease or ventilator-patient asynchrony. If allowed to continue, the increasing lung distension and increasing end-expiratory pressures can have negative hemodynamic and parenchymal effects.[1,3,4,6]

Minute ventilation has an inverse relationship with arterial PCO2. Ongoing monitoring, therefore, is needed to prevent unintended acid-base disturbances. Changes in the patient's spontaneous respiratory rate will naturally change the minute ventilation and may suggest a need to change ventilator settings or mode. In patients using ACMV or PRVC, in which full–tidal-volume breaths are given for every triggered breath, respiratory alkalosis is particularly likely when the spontaneous respiratory rate increases.[1,3,4,6]

The patient-management goal of avoiding complications is an essential addition to the clinical goals of mechanical ventilation. Potential complications of positive-pressure ventilation include decreased cardiac output due to increased intrathoracic pressure, barotrauma, nosocomial infection, acid-base disturbances, increased work of breathing, and unintended hypoventilation. Careful monitoring of ventilator parameters and the patient's vital signs (including oxygen saturation and capnography results) is essential. Sudden changes in a patient's status should provoke a rapid evaluation for problems with the endotracheal tube, ventilator, and ventilator circuit. If those items are intact, changes are more likely to be the result of barotrauma or patient intolerance. Ventilator intolerance should be diagnosed only after excluding physiological or equipment-based causes.[1,6]

Transport Ventilation

Careful patient monitoring is needed during patient transport, whether the patient is being moved between departments or between facilities. Respiratory changes are particularly common during the transport of intubated patients, with changes in PCO2 and pH occurring in 70% to 100% of those patients. Manual ventilation without minute-ventilation monitoring seems to provide the least ventilatory stability. The use of a transport ventilator provides the best ventilatory consistency, but blood-gas changes have been reported when transport ventilators without monitoring of minute ventilation and exhaled tidal volume are used.[2,4,5,12,13]

Although both hyperventilation and hypoventilation occur, unintentional respiratory alkalosis is induced at least 2.5 times as often as respiratory acidosis. Hemodynamic changes probably correlate with blood-gas changes. Hyperventilation can result in air trapping and increased intrathoracic pressures, with resultant hypotension. Myocardial alkalosis is arrhythmogenic and can adversely affect cardiac function. Hypoventilation can cause hypoxemia and acidosis. Cellular acidosis impairs cellular function and causes myocardial irritability.[14-18]

The use of a transport ventilator during any form of transport is advantageous to the patient, but is not a universal practice. The literature does not clearly demonstrate preferences for intrahospital transport, but reports[15,12-16] on other aspects of intrahospital transport suggest that bag-valve-mask–device ventilation without any pressure or volume monitoring is prevalent. Transport programs use a combination of manual ventilation and transport ventilators. Use of a transport ventilator is more likely in patients undergoing interhospital transport than in those transported from the site of injury.[16]

Portable transport ventilators are increasingly available and diverse. These ventilators range from those with basic settings for respiratory rate and tidal volume to much more sophisticated models that can provide both volume-control and pressure-control ventilation with adjustable PEEP, flow, sensitivities, and I:E times. Cost varies with features, but so does clinical flexibility. In fact, the initial purchase price of an advanced transport ventilator exceeds that of all other transport equipment for ventilation, but the cost per patient is similar to that of a bag-valve mask.[19] The American Heart Association recommends that transport ventilators have, at a minimum, a PIP limit, an audible PIP alarm, and inspiratory-time adjustments for adults and children.[17] In general, PIP, minute ventilation, and exhaled tidal volume monitoring are probably indicated.[1,3,4,6] The selection of features needed in a transport ventilator will depend on the agency, patient population, and duration and location of transport episodes. Transport ventilators function as well as ventilators designed for in-hospital use.[18]

Conclusion

Knowledge of ventilation modes and available transport ventilators is only part of the management of critically ill or injured patients. Solid patient assessment, an understanding of the patient's clinical presentation, and the verification of endotracheal-tube placement, together with appropriate mechanical ventilation, are required.

RT

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References

1. Pollack CV. Mechanical ventilation and noninvasive ventilatory support. In: Rosen P, ed. Emergency Medicine Concepts and Clinical Practice. St Louis: Mosby; 1998:21-28.
2. Brower RG, Rubenfeld GD. Lung protective ventilation strategies in acute lung injury. Crit Care Med. 2003;31:S312-S316.
3. [AQ: chapter author. chapter title]. Civetta JM, Taylor RW, Kirby RR, eds. Critical Care. Philadelphia: Lippincott, 1997:[chapter pages].
4. [AQ: chapter author. chapter title]. Zimmerman JL, [two more editors (if there are seven or more) or all editors, if there are six or fewer], et al, eds. Fundamental Critical Care Support. 3rd ed. Des Plaines, Ill: Society of Critical Care Medicine; 2001:[chapter pages].
5. Gervais HW, Eberle B, Konietzke D, et al. Comparison of blood gases of ventilated patients during transport. Crit Care Med. 1987;15:761-763.
6. [AQ: chapter author. chapter title]. Scanlan CL, Wilkins RL, Stoller JK, eds. Egan's Fundamentals of Respiratory Care. New York: Mosby; 1999:[chapter pages].
7. Beker A, Ipekoglu Z, Tureyen K, et al. Secondary insults during intrahospital transport of neurosurgical intensive care patients. Neurosurg Rev. 1998;21:98-101.
8. Braman SS, Dunn SM, Amico CA, Millman RP. Complications of intrahospital transport in critically ill patients. Ann Intern Med. 1987;107:469-473.
9. Martin SE, Agudelo WE, Oschner MG. Monitoring hyperventilation in patients with closed head injury during air transport. Air Med J. 1997;16(1):15-18.
10. Hurst JM, Davis K, Branson RD, Johannigman JA. Comparison of blood gases during transport using two methods of ventilatory support. J Trauma. 1989;29:1637-1640.
11. Perez L, Kolfas E, Wise L. Oxygenation/ventilation of transported intubated adult patients: a national survey of organizational practices. Air Med J. 2000;19:55-58.

12. Cummings RO, Hazinski MF. Emergency cardiac care guidelines. Circulation. 2000;102:SI1-SI384.
13. Miyoshi E, Fujino Y, Mashimo T, Noshimura M. Performance of transport ventilator with patient triggered ventilation. Chest. 2000;118:1109-1115.
14. Hatlestad D. Patient ventilation and oxygenation: a new day dawning. EMS Magazine. 2003;32(1):42-53.
15. Bohn D. Lung salvage and protection ventilatory techniques. Pediatr Clin North Am. 2001;48(3):533-72.
16. Acute Respiratory Distress Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. N Engl J Med. 2000;342:1301-1308.
17. Marelich GP. Protocol weaning of mechanical ventilation in medical and surgical patients by respiratory care practitioners and nurses. Chest. 2000;118:459-467.
18. Grup MJ, Strickland D, Tormey L, et al. Development, implementation, and evaluation of a weaning protocol for patients receiving mechanical ventilation. Am J Crit Care. 2003;12:454-460.
19. Frakes MA. Measuring end-tidal carbon dioxide: clinical applications and usefulness. Crit Care Nurs. 2001;21:23-37.

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